EVALUATION OF RABIES ENCEPHALITIS -PICTORIAL REVIEW

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RABIES ENCEPHALITIS- DISCUSSION

Rabies encephalitis is caused by a type of RNA virus

Transmission by bites of infected rabid dogs, and other wild animals, through inhalation, by contact of infected saliva with an open wound or mucous membrane, and via infected corneal transplants

incubation period -2 to 8 weeks

The viruses are introduced deep into the soft tissues by an animal bite and infect the muscle, possibly through nicotinic cholinergic receptors and may affect the sensory nerve endings

In encephalitic rabies,

brain stem and the cerebrum, particularly the limbic system Involvement , basal ganglia and the thalamus is usually seen late in the disease

patients develop hydrophobia, aerophagia, hypersalivation, hyperirritability, hyperactivity, and priapism.

Neurologic symptoms, such as seizures, agitation, and alternating mood swings, often occur.

Paralytic rabies encephalitis

the medulla and the spinal cord are mainly involved

clinical symptoms of hydrophobia and aerophobia are present in only half these patients

CT SCAN

focal or diffuse areas of decreased attenuation in the basal ganglia, periventricular white matter, hippocampus, and brain stem.

Pontine hemorrhages have also been reported.

Diffuse cerebral edema may be seen in advanced cases.

MRI

Hyperintensity in brainstem, hippocampi, thalami, WM, BG, Paralytic rabies: Medulla and spinal cord hyperintensity on T1- and T2-weighted MR images and not suppresed in Flair

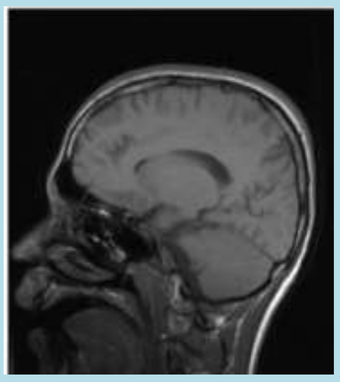
MRI of the brain shows distinct grey matter abnormalities in rabies and is a useful tool when the diagnosis is in doubt.

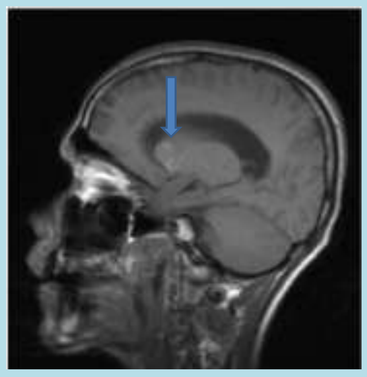
Pathologic specimens of the brain

haemorrhages, necrosis, and surrounding edema in the cerebral gray matter, basal ganglia and the brain stem.

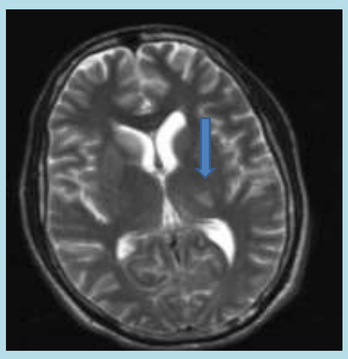
Intraneuronal Negri inclusion bodies inpathologic specimens

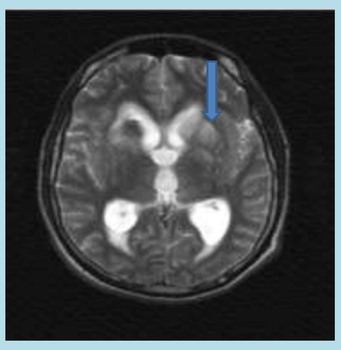
REVIEW AFTER 4 DAYS



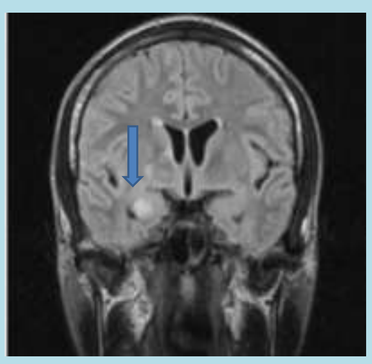


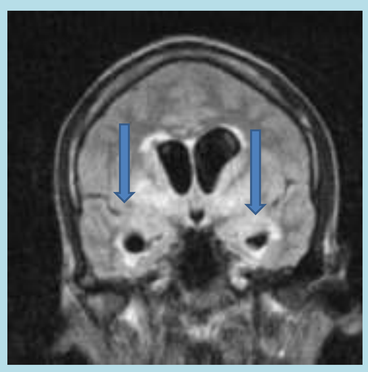
T1W



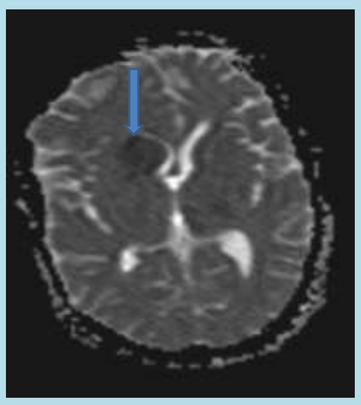


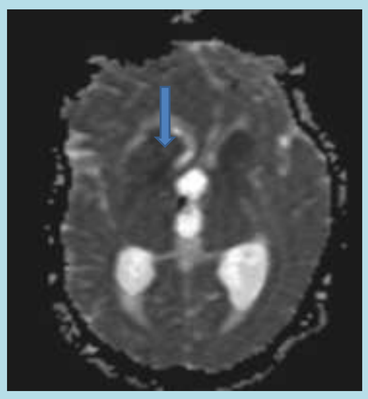
T2W



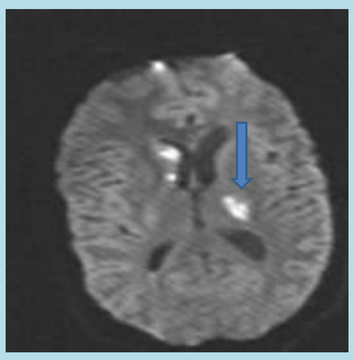


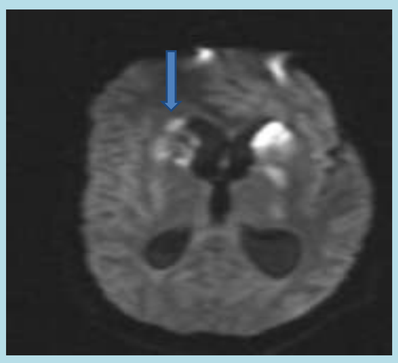
T2 FLAIR



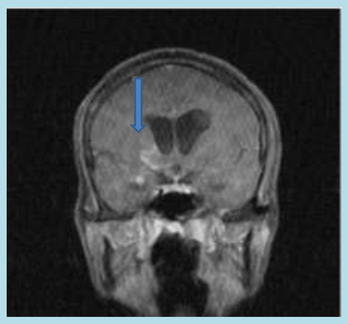


ADC





DW IMAGES



T1 contrast

Clinical history

23 years aged male

H/O dog bite 15 days back on the right upper limb, fever, vomiting, insomnia, photophobia, Diplopia, altered sensorium 5days

Patient had not received anti-rabies immunoglobulin.

CT BRAIN

Hypodensities in Basal Ganglia(bilaterally), pons,midbrain

MRI

T1W images hyperintensity in both hippocampi ,cerebellum, midbrain, pons, periventricular region

T2W images hyperintensity in both hippocampi, cerebellum, midbrain, pons, and periventricular region

FLAIR-not supressed in flair

DW IMAGES- diffusion restriction

T1 CONTRAST

Enhances

On follow up MRI hyperintensity increased &ventricles dilated

DIFFERENTIAL DIAGNOSIS

RABIES	ADEM
caused by RNA virus	Autoimmune-mediated
Predominent grey matter involvement	white matter demyelination of brain and/or spinal cord, usually with remyelination
Hyperintensity in brainstem, hippocampi, thalami, WM, BG, on T1- and T2-weighted MR images	Multifocal punctate to large flocculent FLAIR hyperintensities
	Punctate, ring, incomplete ring, peripheral enhancement

CONCLUSION

Rabies encephalitis and acute disseminated encephalomyelitis have similar clinical presentations but distinct management and prognostic implications. It is thus important to differentiate between the two antemortem. Because of their distinct pathologic abnormalities, MR imaging may be helpful in distinguishing between the two entities.

REFERANCES

AJNR Am J Neuroradiol 22:677–680, April 2001 Manasi Awasthi, Hemant Parmar, Tufail Patankar, and Mauricio Castillo

AJNR Am J Neuroradiol 23:632–634, April 2002Ravi V. Desai, Vivek Jain, Paramjeet Singh, Sunit Singhi, and Bishen Dass Radotra

AJNR Am J Neuroradiol 24:1102–1109, June/July 2003 Jiraporn Laothamatas, Thiravat Hemachudha, Erawady Mitrabhakdi, Pongsak Wannakrairot, and Supoch Tulayadaechanont